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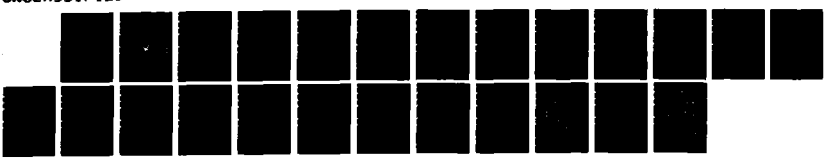

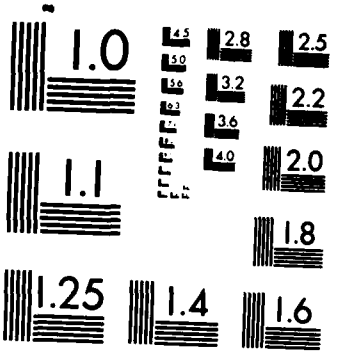

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"SHORT-LATENCY" SOMATOSENSORY EVOKED POTENTIALS DURING EXPERIMENTALLY INDUCED BIODYNAMIC STRESS IN HUMANS

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AVERAGED EVOKED POTENTIALS (EPS) WERE EMPLOYED TO MONITOR THE STRUCTURAL AND FUNCTIONAL INTEGRITY OF HUMAN SOMATOSENSORY PATHWAYS BEFORE AND AFTER EXPERIMENTALLY CONTROLLED EXPOSURES TO IMPACT ACCELERATION. SOMATOSENSORY EPS WERE OBTAINED FROM FIVE RESEARCH VOLUNTEERS USING RECORDING ELECTRODES PLACED ON THE SCALP AND NECK AND OVER ERB'S POINT. THE MEDIAN NERVE OF THE LEFT ARM WAS STIMULATED PERCUTANEOUSLY AT THE WRIST WITH 5/s, .2ms, RECTANGULAR PULSES. CURRENT LEVELS (RANGE 2.5 to 7.0 mA) WERE EQUAL TO THE SUM OF SENSATION PLUS THUMB MOVEMENT THRESHOLDS. THE MAGNITUDE OF THE THUMB MOVEMENT WAS MONITORED THROUGHOUT AN EXPERIMENT. TELEMETERED ELECTROPHYSIOLOGICAL DATA WERE STORED ON MAGNETIC TAPE AND ANALYZED OFF-LINE. IN THE ANALYSIS OF THESE DATA, PARTICULAR ATTENTION WAS PAID TO RELATIVE CONDUCTION TIMES ALONG THE PATHWAY FROM BRACHIAL PLEXUS TO HAND-SOMATOSENSORY CORTEX. THE AMPLITUDE OF THE EARLIER CORTICAL COMPONENTS "N2" AND "P2" (WITH PEAK LATENCIES 19 AND 22 MS, RESPECTIVELY) AND THE ABOSOLUTE LATENCIES OF ALL EP PEAKS WERE ALSO MEASURED AND ANALYZED. THE RESULTS FROM IMPACT ACCELERATION EVENTS UP TO 150 M/S^2 (SLED ACCELERATED FROM ZERO TO 39 MILES PER HOUR IN 5 FEET 2 INCHES) REVEALED NO CLINICALLY SIGNIFICANT ALTERATIONS OF "SHORT-LATENCY" SOMATOSENSORY EPS.

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"SHORT-LATENCY" SOMATOSENSORY EVOKED POTENTIALS DURING EXPERIMENTALLY INDUCED BIODYNAMIC STRESS IN HUMANS

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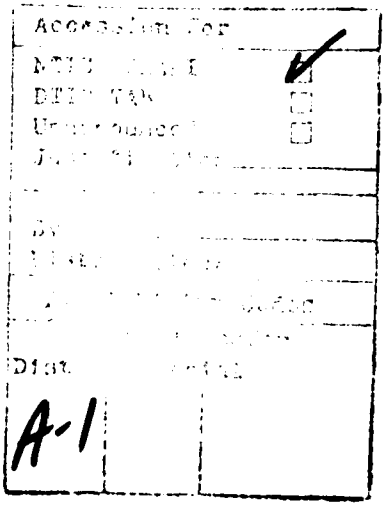
ABSTRACT

Averaged evoked potentials (EPs) were employed to monitor the structural and functional integrity of human somatosensory pathways before and after experimentally controlled exposures to impact acceleration. Somatosensory EPs were obtained from five research volunteers using recording electrodes placed on the scalp and neck and over Erb's point. The median nerve of the left arm was stimulated percutaneously at the wrist with 5/s, .2 ms, rectangular pulses. Current levels (range 2.5 to 7.0 mA) were equal to the sum of sensation plus thumb movement thresholds. The magnitude of the thumb movement was monitored throughout an experiment. Telemetered electrophysiological data were stored on magnetic tape and analyzed off-line.

In the analysis of these data, particular attention was paid to relative conduction times along the pathway from brachial plexus to hand-somatosensory cortex. The amplitude of the earliest cortical components "N2" and "P2" (with peak latencies 19 and 22 ms, respectively) and the absolute latencies of all EP peaks were also measured and analyzed. The results from impact acceleration events up to 150 m/s^2 (sled accelerated from zero to 39 miles per hour in 5 feet 2 inches) revealed no clinically significant alterations of "short-latency" somatosensory EPs.



INTRODUCTION

Head and neck injuries which result from accidental impact are of major significance in both the civilian and military communities (National Safety Council, 1979; Reader, 1979). At the Naval Biodynamics Laboratory (New Orleans), a major research program is underway to investigate the biomechanical, physiological and behavioral effects of indirect impact of the head and neck under carefully controlled experimental conditions. The ultimate goal of this research is the development of mathematical and engineering models which predict injuries of the head and neck. Part of this modeling effort involves the use of short-latency somatosensory evoked potentials (SSEPs) recorded from both Rhesus and humans as possible indicators of central nervous system dysfunction.

For impact accelerations in the -X direction (frontal impact) preliminary results in Rhesus suggest that increases in the peak latencies of cervical and cortical evoked potentials may serve as indicators of impending damage to the central nervous system (Saltzberg & Burton, 1979; Berger & Weiss, 1982; Weiss & Berger, (in press)). Specifically, significant transient increases in the peak latency of cervical and cortical SSEPs occurred above 600 m/s^2, while neuropathological damage occurred at levels of impact acceleration exceeding 720 m/s^2 (Unterharnscheidt, 1982). Transient reduction in the amplitude of the cortical SSEPs occurred as a linear function of the level of -X acceleration with 50% to 100% reduction at accelerations above 600 m/s^2.

The present paper reports the first attempts to extend these results to include SSEP data recorded non-invasively from humans experiencing moderate levels of experimentally controlled impact acceleration.

METHOD

Subjects (Ss) were five healthy enlisted males (age range 18 to 22 years) assigned to the Naval Biodynamics Laboratory. All subjects were recruited, evaluated and employed in accordance with procedures specified in Secretary of the Navy Instruction 3900.39 and Bureau of Medicine and Surgery Instruction 3900.6. Subjects were given cardiovascular, pulmonary, skeletal and other examinations to ensure their capability to serve in possibly hazardous environmental research (Thomas, et al., 1978). A physician attended all acceleration experiments, and emergency medical care facilities were immediately available. Subject's heart rate and EKG were monitored continuously during each experiment.

An experiment involved approximately 30 to 40 minutes of recording SSEP and inertial data before, during and after impact acceleration in the -X (frontal impact) direction (Thomas, et al., 1974; 1975). Subjects sat upright on a 2,494 kg sled which, at the moment of impact, was accelerated along a level track by a Bendix 12-inch HYGE® system with a piston stroke ranging from 0.7 m at 20 m/s^2 to 1.62 m at 150 m/s^2. The acceleration phase lasted less than 150 ms with onsets ranging from 472 m/s^2 to 5356 m/s^2. This resulted in a velocity of up to 17.6 m/s (39 mph). The deceleration phase lasted many seconds but was negligible (mean force of friction 2 m/s^2; range .7 m/s^2 to 3 m/s^2). The subject restraint system consisted of a three inch wide dacron shoulder strap with lap belt and inverted V. Arm restraints above the elbow were used and the head and neck were freely moveable. As indicated in Fig. 1, SSEP data were obtained first during one seven-minute recording period, before the impact event, while the subject sat quietly with restraints in place but loose. SSEP data were then obtained also during two four-minute periods, one just before and one just after impact, while the subject sat

quietly with restraints tightened. SSEP recording was not stopped during the sled movement nor during the period of approximately three to seven minutes after impact when restraints were being loosened and inertial gear was being removed. This was followed by a second uninterrupted seven-minute period of recording with the subject quietly resting in loose restraint.

Subjects were exposed to increasing acceleration levels in the order 20, 30, 40, 60, 80, 100, 120, 130, 140, and 150 m/s^2. SSEPs were recorded during the 20, 100, and 150 m/s^2 exposures except for one subject for whom SSEPs were obtained at all acceleration levels. During five months of testing, ambient air temperature (AAT) near the pre-impact sled position varied from 59°F to 80°F (mean 74°F). AAT was lower near the post-impact sled position by as much as 12°F on one occasion but generally by about 3 to 5°F. When necessary, subjects were kept warm with sheets, and oral temperature was recorded at the beginning and end of each experiment.

Stimulating and recording cup electrodes (Ag and Ag/AGCl, respectively; 1 cm diameter) were affixed with collodion soaked gauze after the skin was prepared by rubbing with pumice paste and alcohol soaked cotton. Conductive jelly was inserted by blunted needle and syringe through a hole in the base of the cup electrode (impedances for stimulating and recording electrodes less than 5k and 2k ohms, respectively). The left median nerve was stimulated percutaneously at the wrist (5 Hz; .2 ms rectangular pulse; isolated from system ground; constant current). The level of stimulus current was set for each sled run at the sum of sensation plus thumb-movement thresholds (mean 4.8 mA; range 2.6 to 8.7 mA). The anode was placed one cm proximal to the flexion crease of the wrist with the cathode 3 or 4 cm proximal to the anode. Medio-

laterally, the two stimulating electrodes were placed between the tendons of the palmaris longus and flexor carpi radialis. The subject was grounded to the system ground only on the proximal, volar surface of the left forearm. The elbow, lower arm, wrist, hand and fingers were immobilized by taping them into a fiberglass posterior-surface cast. Recording electrodes were placed: (1) overlying the clavicle anterior to the left Erb's Point, (2) overlying the iliac crest of the right hip, (3) midline on the neck 4 cm caudal to inion, (CII), (4) at Fz, and (5) at C4' (2 cm posterior to C4).

Four channels of EEG (Table 1) recorded by means of impedance-matching amplifiers attached to the subject only inches from the electrode cups, and from there by means of low-noise cables into high-gain amplifiers mounted on the sled. Data were telemetered to an instrumentation "block-house" where the EEG (50 to 1500 Hz; -3dB) was recorded in both multiplex and FM formats on separate Ampex® FR2000A tape recorders along with stimulus markers and precision time code. SSEPs were monitored on-line from tape playback during an experiment using a Nicolet® 1172 Signal Averager.

Off-line, two SSEP averages of 1000 trials each were obtained from each of the 7-minute (loose restraint) recording periods, and two SSEP averages of 300 trials each were obtained from each of the 4-minute (tight restraint) recording periods by means of a Nicolet® MED-80 Computer. Data from the interval extending from 10 seconds pre-impact to 30 seconds post-impact were excluded from the averages. The duplicate traces in each condition aided in confirmation of peak selection. For statistical analyses, peak latency data were chosen by cursors placed on traces with N=2000 (loose restraint) and 600

TABLE 1: RECORDING CONFIGURATION

CHANNEL	+	-	GAIN	PEAKS
1	input left Erb's point	input C4'	100K	BP
2	Fz	right hip	100K	P9, P11, P13, P14
3	Fz	CII	100K/200K	A, B
4	Fz	C4'	100K/200K	N1, N2, P2

Recording configuration and source of identified peaks.

trials (tight restraint). Individual trials of excessively high amplitude were excluded from averages. Most waves were labeled with indication of polarity (P or N) and mean peak latency during the 20 m/s^2, pre-impact, loose restraint period (Figure 2 and Table 2). Exceptions were BP, a negative-going near-field wave originating in the brachial plexus, the A and B waves of Chiappa, et al. (1980), and the N1, N2, and P2 waves recorded from Channel 4. Peak latencies of the components listed in Table 2 were subject to individual BMDP P2V ANOVAs (Dixon, et al., 1981). In addition, P2V ANOVAs were performed on the peak latency differences listed in Table 3, as well as the N2-P2 peak-to-trough amplitude.

All P2V ANOVAs were conducted using the option for orthogonal decomposition of within-subject factors and errors (orthogonal) and the option for testing the efficacy of pooling error components (symmetry). Linear and quadratic error components were pooled only when the respective symmetry assumptions appeared unviolated ($P > .25$), following the recommendation of Winer (1971). Linear and quadratic components of each factor were separately analyzed based on the presumption that effects would be primarily linear.

RESULTS

Figure 2 and Table 2 illustrate the temporal relations among the ten peak latency measures. Figure 2 shows averaged SSEP traces for one subject. The averaged waveforms were similar to those illustrated previously by Desmedt and Cheron (1980; 1981) and by Chiappa, et al. (1980). Table 2 shows the baseline means and standard deviations of the SSEP peaks for all five Ss, derived from the loose-restraint, pre-impact recording period of the 20 m/s^2 experiments. For all Ss, P9 preceded BP and P11 preceded A. A similar temporal ordering occurred for the other peaks with one exception: P13 preceded B in one of the five Ss. These two peaks, B and P13, were the only two with overlapping 95%

TABLE 2: PEAK IDENTIFICATION

Peak:	P9	BP	P11	A	B	P13	P14	N1	N2	P2
(Latency) (ms)	8.95	9.80	11.39	11.98	13.30	13.52	14.27	17.69	19.26	22.59
s.d. (ms)	.16	.23	.21	.18	.16	.20	.22	.48	.57	1.06

Five subject means and standard deviations for the latency of each of the peaks.

TABLE 3: INTER-PEAK CONDUCTION TIMES

Peaks:	P9 BP	P9 P11	BP P11	P11 A	A B	A P13	B N2	P13 N2	N2 P2
Conduction Time (ms)	.85	2.44	1.59	.66	1.32	1.54	5.96	5.74	3.33
s.d. (ms)	.16	.08	.13	.15	.28	.37	.56	.69	.52

Five subject means and standard deviations for the interpeak conduction times.

confidence intervals, so uncertainty in temporal ordering would be expected.

In the analysis of variance (ANOVA) performed on the individual peaks of Table 2, data for P9, P11, A, B, P13 and P14 obtained during the tightly restrained interval (Fig. 1) were not included. These data were especially contaminated with high amplitude artifacts (including EKG). Therefore, SSEPs computed from the artifact-free portions of the data suffered from poor signal-to-noise ratios (due to decreased sample size) so that identification of these individual peaks was not possible. As a consequence, the ANOVA performed on the interpeak conduction times (Table 3) used only data obtained during the loose restraint intervals (Fig. 1).

The signficant results from the ANOVA are listed in Table 4. Conduction time from P13 to N2 decreased linearly from 5.85 ms at 20 m/s^2 to 5.72 ms at 150 m/s^2 as did the P9 to BP conduction time (from .95 ms at 20 m/s^2 to .80 ms at 150 m/s^2). Since successively higher levels of acceleration were used in sequence many days or weeks apart, these effects could be due to an overall temporal trend. Alternatively, anticipation of higher levels of acceleration may effect SSEP measures.

Pre-impact versus post-impact comparisons showed statistically significant changes in three peak latencies. Peak BP was delayed by 60 μs while N2 and P2 occurred sooner by 65 and 112 μs respectively. The change in BP may be a mechanical artifact produced by the restraint system which passed close to or over the Erb's point electrode. If N2 and P2 are indeed cortical in origin (Desmedt & Cheron, 1981) then the small latency decrease may well reflect an arousal influence.

Of special interest are those changes which show a significant pre-impact to post-impact effect which varies with the level of acceleration. The

TABLE 4: SUMMARY OF SIGNIFICANT ANOVA RESULTS

FACTOR	MEASURE	SIGNIFICANCE LEVEL	PERCENTAGE* SUBJECT VARIATION
Acceleration	P9 to BP	.06	48.4%
(Linear	P13 to N2	.05	4.3%
Component)			
Time	BP	.04	8.8%
(Before or	N2	.008	1.5%
after sled	P2	.03	1.8%
movement)			
Acceleration	BP to P11	.06	12.2%
(Linear	P14	.05	34.5%
Component)	N2 - P2 (amp)	.005	5.1%
X			
Time			
Acceleration	N2 to P2	.06	0.6%
(Quad. Com-			
ponent			
X			
Time	N1	.05	3.1%
Restraint	N2 - P2 (amp)	.01	2.9%

*Ratio of the effect mean square to mean square for subjects.

latency measures which demonstrated such an effect were the BP to P11 conduction time and the P14 latency. Again, the BP effect may be artifactual, ranging from a 32 μs increase at 20 m/s^2 to a 17 μs increase at 150 m/s^2. The linear shift in P14 ranged from a 240 μs increase at 20 m/s^2 to a 24 μs decrease at 150 m/s^2. The relative shift in these latencies was of the same order, ranging up to a 15% to 20% increase.

The one amplitude measure which was analyzed, N2 to P2 peak-to-trough amplitude (2.43 $\pm$ .90 μV baseline value) showed two statistically significant effects. First, the amplitude was reduced (2.17 μV) when the Ss were tightly restrained as compared to the loosely restrained condition (2.31 μV). Second, the N2-P2 amplitude changed post-impact as compared to pre-impact. This was a linear effect relative to the level of acceleration with amplitude increasing by .11 μV at 20 m/s^2 by .02 μV at 100 m/s^2 and decreasing by .21 μV at 150 m/s^2. Taking the 20 m/s^2 pre-impact, loosely restrained value of 2.43 μV as a reference, this effect ranged from a 5% increase to a 9% decrease in amplitude.

There were no satistically significant effects for oral temperature taken before and after each impact acceleration experiment, nor for level of stimulation (sensation threshold, movement threshold, and sum of sensation and movement thresholds).

DISCUSSION

The present study is the first one in which SSEPs have been recorded from humans undergoing -X impact acceleration. As such, the study was exploratory in nature and directed towards answering two specific questions. First, and

most important, were any clinically significant modifications in somatosensory pathway function noted as a result of exposure to moderate levels of acceleration? Second, what modifications in experimental procedures are recommended for future work?

In response to the first question, there were several statistically significant and important findings in this study, but none of them appear to have clinical significance. This is due to the fact that none of the measured changes were large compared to variation between and within subjects. Between-subject standard deviations ranged from 80 to 1060 μs (Tables 2 and 3) while within-subject standard deviations (derived from a separate, five-day evoked potential reliability study) ranged from about 200 to 340 μs. In clinical settings, variations of two to three or more standard deviations from group normative means are required before clinical significance is claimed. In the present experiment, the observed changes were well within one standard deviation of the baseline results, with the exception of the shift in the P14 latency after impact which showed the greatest shift (240 μs) at the lowest level of acceleration. From Table 2 the standard deviation of P14 is 220 μs, which is slightly less than the observed increase in latency. Even in this case, the effect is too small to be of clinical note, in addition to its occurrence at the most inoccuous level of acceleration.

It is important to note that our measurement and analysis technique is very sensitive and any substantial changes in the SSEP of potential clinical importance would not be overlooked. These measurements show that -X acceleration levels up to 150 m/s^2 do not alter the SSEP in any clinically significant way.

Concerning the second question of modifications in our experimental procedures, there are two important findings. The tight restraint system influenced results both by reducing the amplitude of the N2-P2 cortical component and by introducing excessive noise into our recordings. In particular, the CII and right hip electrodes were particularly sensitive to the tightening of the restraint system. Redesigning our recording montage with this in mind should reduce the noise problem. Finding a good non-cephalic reference is the major requirement for solving this problem and will also enable us to resolve some of the near-field vs. far-field ambiguities of our current montage. Monitoring of restraint tension is also suggested as a means of controlling for the effects of a tightened restraint.

Though this analysis of the first SSEPs obtained from human volunteers undergoing experimentally controlled impact acceleration has resulted in no clinically significant findings, SSEP changes were reliably measured. The analytical techniques appear promising and with refinement and modification, we hope to improve upon them. This will ultimately provide useful input to the development of head and neck injury models.

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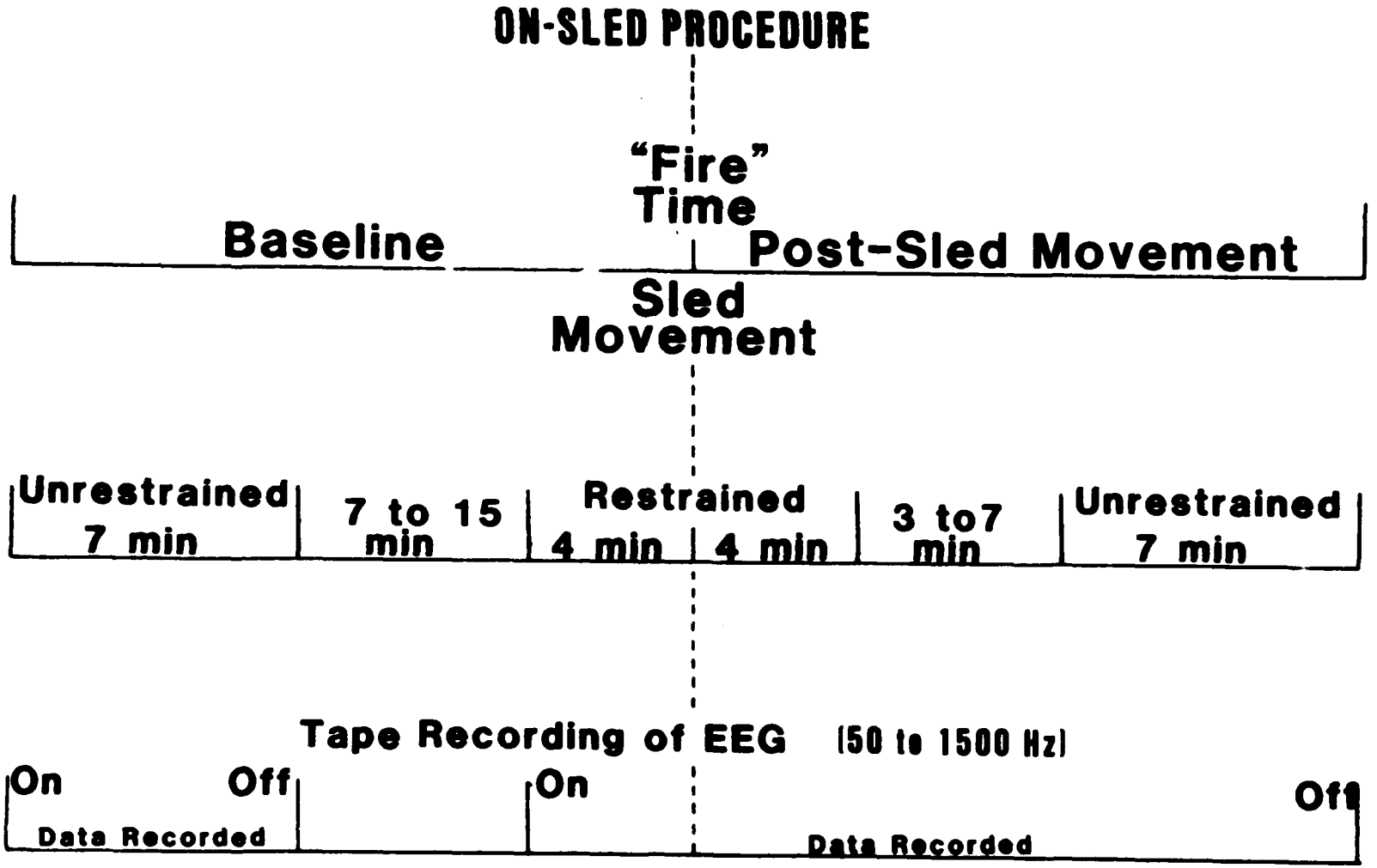
Figure 1: Schematic illustration of the pre-impact and post-impact data recording and safety restraining sequence.

Figure 2: Sample SSEP from a human volunteer subject, indicating the peaks selected for detailed analysis (also see Table 1).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Baseline
Post-Sled Movement
Sled Movement
Unrestrained 7 min
7 to 15 min
Restrained 4 min
4 min
3 to7 min
Unrestrained 7 min
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On
Off
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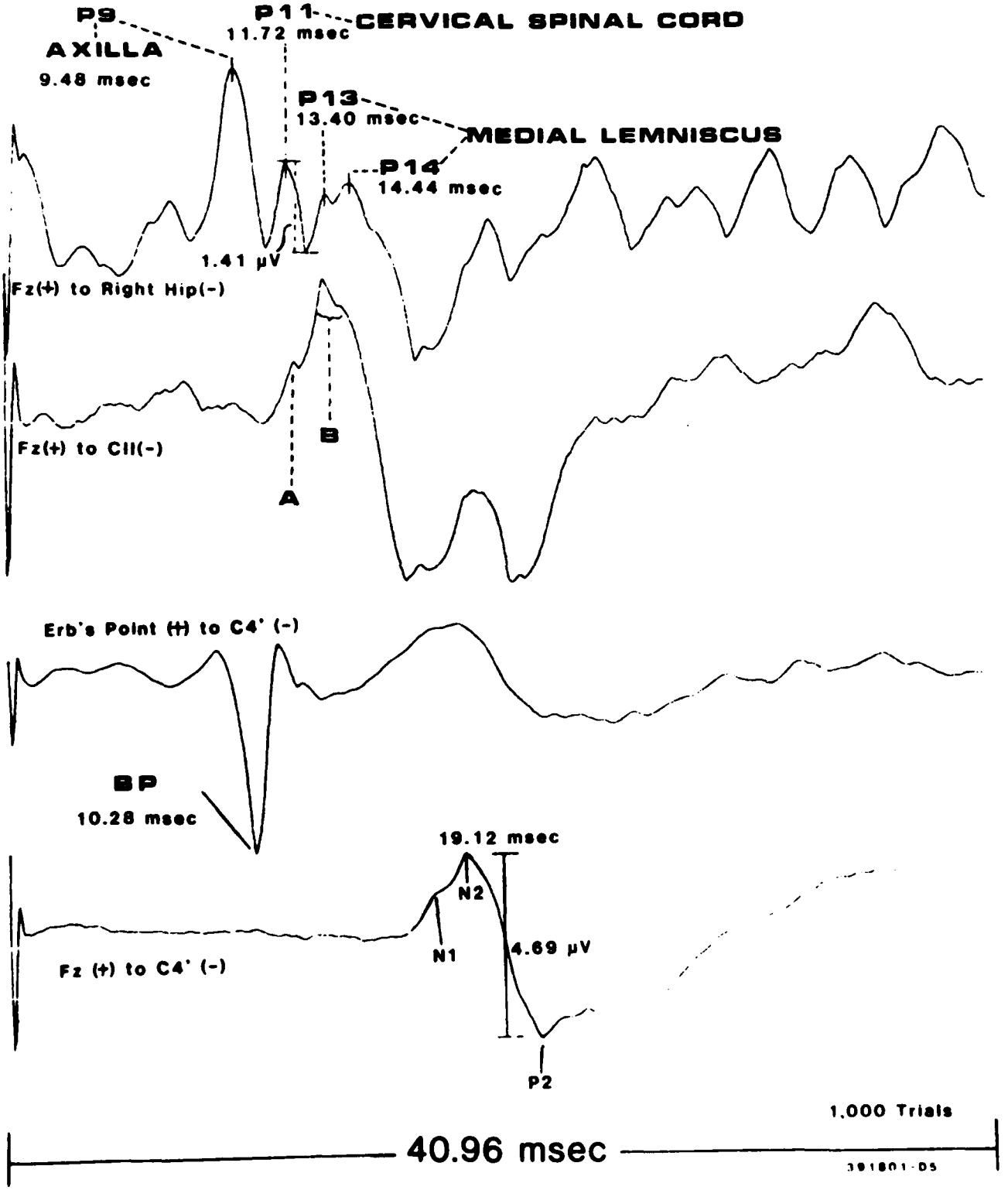
HUMAN SSEP - PEAK IDENTIFICATION



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